cereus indicate that streptomycin is adsorbed by bacterial cells, all or nearly all of the adsorption occurring at the cell surface. There was no demonstrable difference in adsorption between susceptible and resistant strains of these organisms. The affinity of streptomycin for bacterial cells is greatly reduced in the presence of NaCl. Evidence is presented indicating that the persistent action of streptomycin *in vivo* after the blood level falls below a detectable value is not related to a slow desorption of streptomycin from the adsorbed state on bacterial cells.

16394 P

An Outbreak of Equine Encephalomyelitis, Eastern Type, in Southwestern Louisiana.

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From May to October of 1947 an outbreak of encephalomyelitis occurred among horses and mules in southwestern Louisiana. Thousands were implicated with approximately 3,713 deaths.[1] The disease began in Iberia Parish, spread west to Texas and northwest to DeSoto Parish. Because the eastern type of equine encephalomyelitis virus was recovered from human cases that occurred coincidentally, a preliminary report is submitted.

The eastern equine virus was first isolated from horses in 1933,[2,3] and from children in Massachusetts during 1938.[4,5] Antibodies were found in the serum of an adult in 1939.[6] All cases of the eastern type were thought to be confined to the Atlantic coastline until 1940[7] and 1941[8] when it was recovered from horses in both Alabama and Texas. Subsequently equine cases have been reported in all of the Gulf States;[9] also in Missouri,[9] Michigan,[10] Mexico,[11] Brazil[12] and Panama.[13] Besides the Massachusetts outbreak the only human cases ascribed to the eastern strain were 3 in Texas[14,15] that showed neutralizing antibodies in the serum and 1 in Louisiana from whom the virus was recovered in 1946.[16]

In the present outbreak all human cases except one were in children from 7 months to 15 years of age. The one fatal adult case

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TABLE I.
Results of Serum Neutralization Tests.

Source of sera	E.E.E. virus			W.E.E. virus		
	No. tested	No. positive	% positive	No. tested	No. positive	% positive
Human						
Died	6	1 pos. 2 wkly*	50	6	0	0
Recovered	5	3	60	5	2 wkly	40
Contacts	22	4	18.1	16	0	0
Neighbors	23	0	0			
Horses and Mules						
Recovered	11	8	72.7	11	2 pos. 1 wkly	27.2
Sick	4	4	100	4	1 pos. 3 wkly	100
Normal	14	9	64.2	12	1 "	8.3
Cows	5	2 wkly	40.0	3	2 "	66.6
Chickens	105	2 pos. 2 wkly	3.8	88	19	21.5
Ducks	8	0	0	8	0	0
Geese	8	1 wkly	12.5	8	0	0
Turkeys	3	0	0	3	0	0
Pigeons	35	1 wkly	2.8	30	0	0
Pig	1	0	0	1	0	0
Dogs	3	1	33.3	3	1	33.3

* wkly = weakly positive.

was aged 74. The patients were from rural or small town areas; many mosquitoes were observed and most of the houses were unscreened.

Case history of E. R. D. The following typical case history is submitted. A 2-year-old white girl from Maurice, Louisiana, became restless, irritable, drowsy, febrile and had several generalized convulsions. The following day a lumbar puncture revealed a lymphocytic pleocytosis. She was taken to New Orleans Charity Hospital, where examination revealed an acutely ill, febrile, comatose child, having frequent convulsions. There was stiffness of neck, back, and upper and lower extremities. Despite chemo-and supportive therapy the patient became progressively worse with almost continuous convulsions, until death occurred on the fifth day of illness. The virus of eastern equine encephalomyelitis was recovered from the brain.

Laboratory findings. Fresh brain material was obtained from 6 fatal equine cases, 3 of them through the courtesy of the State Veterinary Department and from 8 human fatalities through the courtesy of the Louisiana State Health Department and the Tulane Service of Charity Hospital, New Orleans.

Brain suspensions were inoculated intracerebrally and intraabdominally into guinea pigs and 10-day-old white mice with recovery of a virus from 2 human and 3 equine tissues. It could be passed through Mandler and Seitz filters, was lethal for mice, guinea pigs and hamsters in 48 hours and for embryonated eggs in 24 hours. The mice showed a tremor, drowsiness, hyperexcitability followed by convulsions. The guinea pigs exhibited a temperature of 106°C and a rhythmic trotting motion of all 4 extremities. Identification was accomplished by means of the neutralization test in mice. All new strains were neutralized by immune serum of the eastern equine virus and not by that of the western and the St. Louis encephalitis. Hyperimmunized guinea pigs were inoculated intracerebrally. Those animals immune to the eastern equine virus remained alive while the normal controls and guinea pigs immune to the western strain died in 48 hours. Serum from a recovered guinea pig given human brain material contained complement fixing antibodies against the eastern but not the western equine antigen.

Neutralization tests against the eastern and

western equine viruses were performed with the sera of equine and human recovered cases and their contacts and also with sera from domestic animals found on the farms of fatal cases. The results are given in the Table I. Besides the positive results with the human and horse sera only 2 chickens and 1 dog showed strong antibodies for the eastern virus. The sera of 2 chickens, 1 goose, 1 pigeon and 2 cows were only weakly positive. Many of these tests were performed with one of the newly isolated human strains of virus. It was of interest that 25.9% of the unvaccinated horses, 18.1% of the chickens and 1 dog had antibodies for the western equine virus, while the sera of 2 cows and 2 people were weakly positive.

The histopathology in the horse brains showed focal and diffuse gliosis, perivascular round-cell infiltration and edema. The motor neurons showed chromatolysis with cytolysis and phagocytosis by the glial cells. Passage of a human strain into guinea pigs produced severe neuron degeneration and mild perivascular infiltration with edema.

Arthropods were collected from the different areas where encephalitic cases occurred. A later report will be presented as well as a more detailed epidemiological account of this outbreak. The clinical cases will be described by Dr. J. Syverton of Louisiana State University.

Summary. The virus of eastern equine encephalomyelitis was recovered from the brains of both human and equine fatal cases occurring in an outbreak of encephalomyelitis in southwestern Louisiana. Positive neutralization tests to this virus were obtained with sera of 2 chickens, 1 dog, and 31 human and equine cases and their contacts, while those of 2 chickens, 1 goose, 1 pigeon and 2 cows were only weakly positive. Antibodies to the western equine virus were found in the sera of 1 dog, 8 horses and 19 chickens and to a very slight degree in the sera of 2 humans and 2 cows.

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Cold Hemagglutinin in Chinese Kala Azar.

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Rose[1] observed high titers of cold hemagglutinin in two cases of kala azar and concluded that such agglutinin developed as a result of the parasitic infection. This observation has been studied in order to determine its possible clinical significance in this disease.

Method and Material. Collection of blood specimens and the titration of the cold hemagglutinin content in fresh serums were carried out strictly according to Rose's method.[1] In nearly all of the tests reported in this paper, group O erythrocytes from one of us, Hou, were used. The final dilution of the serum in the last tube showing fine granular clumpings was recorded as the titer of each test. The serums studied included specimens from 68 proven kala azar patients, 62 normal individuals, and 12 atypical pneumonia cases.

Results. The results of the above mentioned investigations are summarized in Table I.

a. *Normal controls.* Cold hemagglutinin with titers varying from 1:4 to 1:32 was present in 56.4% of 62 normal individuals, but absent in the remaining 43.6%. The highest titer recorded in our normal controls was 1:32. Therefore in the present study only titers above 1:32 have been considered significant.

b. *Primary atypical pneumonia cases.* This

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